Viral encephalitis in adults

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INTRODUCTION

Viral infections of the central nervous system (CNS) result in the clinical syndromes of aseptic meningitis (infection of the meninges) or encephalitis (infection of brain parenchyma) [1-3]. The incidence of these infections is difficult to determine as many cases are unreported, the diagnosis may not be considered, or a specific viral etiology is never confirmed. Molecular diagnostic testing has significantly improved the detection of viral pathogens from cerebrospinal fluid (CSF) [4]. Clinicians should be familiar with the clinical manifestations, diagnostic techniques, and therapeutic options for encephalitis [5].

This topic will review the major distinguishing features between aseptic meningitis and viral encephalitis, as well as the viral etiologies that can cause encephalitis. Topic reviews that discuss the clinical manifestations, diagnosis, and pathogens associated with aseptic meningitis in adults and viral encephalitis in children are found elsewhere. (See "Aseptic meningitis in adults" and "Acute viral encephalitis in children: Clinical manifestations and diagnosis" and "Acute viral encephalitis in children: Treatment and prevention" and "Acute viral encephalitis in children: Pathogenesis, epidemiology, and etiology".)

MENINGITIS VERSUS ENCEPHALITIS

The presence or absence of altered mental status is the critical distinguishing feature between encephalitis and meningitis. Patients with meningitis may be uncomfortable, lethargic, or distracted by headache, but their cerebral function remains normal. Seizures causing transient postictal confusion can be seen with meningitis, but confusion typically resolves within hours.

In contrast, a required criterion for encephalitis is the presence of altered mentation, defined as decreased consciousness, lethargy, or personality change lasting at least 24 hours with no alternative cause identified [6]. Other criteria for the diagnosis of encephalitis are provided in the table (table 1).

However, the distinction between the two entities can be challenging since some patients may have both a parenchymal and meningeal process with clinical features of both. In these cases, the term meningoencephalitis is used to recognize the overlap. The importance of distinguishing between viral encephalitis and aseptic meningitis relates to the fact that the most common infectious causes of each syndrome differ, as does the prognosis [7]. (See "Aseptic meningitis in adults".)

VIRAL VERSUS POSTINFECTIOUS ENCEPHALITIS

Viral encephalitis can be either primary or postinfectious (ie, encephalitis occurring as a sequela of prior viral infection) [8].

- **Primary viral encephalitis** – Primary infection is characterized by viral invasion of the central nervous system (CNS). Neuronal involvement can be identified on histologic examination, which may show inclusion bodies on light microscopy or viral particles on electron microscopy. The virus can often be detected in brain tissue by immunohistochemistry, nucleic acid amplification, or, less frequently, culture.

- **Autoimmune (postinfectious or paraneoplastic) encephalitis** – Preceding infections may play a role in triggering autoimmune encephalitis. In the case of acute disseminated encephalomyelitis (ADEM), a postinfectious condition, white matter demyelination is common, however virus cannot be detected or recovered [8-10]. Anti-NMDA receptor encephalitis is an autoimmune condition associated with the development of neuronal cell-surface antibodies, which can be triggered by antecedent viral infections, particularly herpes simplex encephalitis [11-13]. These conditions are discussed in detail elsewhere. (See "Acute disseminated encephalomyelitis (ADEM) in adults" and "Autoimmune (including paraneoplastic) encephalitis: Clinical features and diagnosis".)

Differentiating viral versus postinfectious encephalitis has important management implications, as the latter may respond to immunomodulating agents. When the diagnosis is not clear, a multidisciplinary approach with consultation by specialists in infectious diseases and neuroimmunology is recommended [7]. (See 'Viral pathogens' below.)

EPIDEMIOLOGY

Etiology often unidentified — The frequency of identifying a specific agent in patients with encephalitis is variable and depends upon the population studied and the mode of testing. As examples, various large studies of patients with encephalitis reported a cause identified in 28 to 79 percent of patients [14-16]. In contemporary studies, autoimmune encephalitis accounts for 16 to 25 percent of cases initially thought to be infectious in nature [14,17-19]. (See "Autoimmune (including paraneoplastic) encephalitis: Clinical features and diagnosis".)

Viral pathogens — Of patients with an identified cause of encephalitis, viral pathogens are most common (36 and 82 percent of all diagnoses in two separate studies) [15,16]. A wide variety of different viruses can infect the central nervous system (CNS). (See 'Meningitis versus encephalitis' above.)

The most common cause of sporadic encephalitis is herpes simplex virus (HSV) type 1 [14,20]. Other common viral pathogens in both the United States and internationally include varicella-zoster virus (VZV), West Nile virus, and enteroviruses [7,14,21]. (See 'Epidemiologic clues' below.)

Epidemiologic clues — In some cases, the cause of viral encephalitis is suggested by the exposure history (table 2).

- **Seasonality** – While HSV encephalitis can occur year-round, other infectious causes of encephalitis have seasonal associations (table 2). Arboviruses (eg, eastern equine, western equine, St. Louis, La Crosse virus, and West Nile virus) cause disease when mosquitoes are active, while exposure to outdoor areas with high tick populations might suggest a tick-borne infection (eg, Powassan virus). Other nonviral tick-borne etiologies include Ehrlichiosis, Lyme disease, and Rocky Mountain spotted fever. (See "Epidemiology and pathogenesis of West Nile virus infection" and "Arthropod-borne encephalitides" and "Epidemiology, clinical manifestations, and diagnosis of Rocky Mountain spotted fever" and "Arthropod-borne encephalitides", section on 'Specific viruses'.)

- **Geography** – Viral causes of encephalitis vary geographically. As examples, Powassan virus, transmitted by *Ixodes* ticks, is endemic to eastern Canada and the north central and northeastern United States [22,23]. Eastern equine encephalitis virus occurs in the northeastern region, Atlantic and Gulf coasts, and Great Lake Region of North America but is also found in the Caribbean, South America, and Central America [24]. St. Louis encephalitis virus infection occurs in North America, predominantly throughout the midwestern and southern United States. Tick-borne encephalitis virus, also transmitted by

Ixodes ticks, is one of the most common causes of encephalitis in Europe and Asia [25]. West Nile virus is the most common vector-borne cause of viral encephalitis in the United States [26,27]. West Nile virus first appeared in the United States in 1999, although it had already been established in Africa, Asia, and Europe, and then spread rapidly across the country. The incidence of other arboviruses varies significantly based upon geographic region. (See "Clinical manifestations and diagnosis of West Nile virus infection" and "Arthropod-borne encephalitides".)

- **Animal exposure** – Specific animals are associated with various viral causes of encephalitis. Nipah virus encephalitis, which has appeared in Malaysia, Singapore, the Philippines, Bangladesh, and India, may be associated with exposure to pigs, fruit bats, or horses [28-32]. Human infection with Hendra virus, which is seen in Australia, results from direct contact with infected horses. Encephalitis due to avian influenza virus has been reported after swimming in natural bodies of water with large duck populations [33]. Lymphocytic choriomeningitis virus is a human-acquired zoonosis caused by a rodent-borne arenavirus, which can be transmitted through exposure to secretions of mice, rats, and hamsters. Variegated squirrel bornavirus 1 has been identified as a cause of encephalitis in three German breeders of variegated squirrels; the virus was also detected in a squirrel from the breeding population of one of the patients [34]. (See "Nipah, Hendra, and other henipaviruses" and "Avian influenza: Epidemiology and transmission" and "Aseptic meningitis in adults".)

  Prior history of an animal exposure or bite may also suggest the possibility of rabies encephalitis; however, the absence of such a history does not eliminate the possibility of rabies. In the United States, human rabies is most commonly transmitted through bites from wildlife, particularly bats, whereas internationally, dog bites are the leading cause of infection. Infections associated with domesticated and exotic animals [35,36] have also been reported. (See "Clinical manifestations and diagnosis of rabies".)

CLINICAL MANIFESTATIONS

Signs and symptoms — Signs and symptoms of encephalitis reflect brain inflammation with associated neurologic dysfunction. Clinical criteria supporting a diagnosis of encephalitis are listed in the table (table 1). As many of the clinical findings are nonspecific, exclusion of other causes

(eg, metabolic disease, brain ischemia, toxin ingestion) is necessary.

Altered mentation lasting for at least 24 hours is universally present and may range from subtle cognitive deficits to obtundation. New psychiatric manifestations may include psychosis, agitated delirium, or catatonia.

Clinical criteria for the diagnosis of encephalitis

Table 1

While altered mental status is a requisite finding, other signs or symptoms are variably present. Fever, while common, may be absent in older or immunocompromised individuals. Seizures may be obvious or may be subclinical. New onset refractory status epilepticus following a febrile illness may mimic encephalitis but be indicative of febrile infection-related epilepsy syndrome [37]. Meningeal signs and by extension cerebrospinal fluid (CSF) pleocytosis are frequently, but not universally, present [17].

Clues on physical examination — Clinical presentation of encephalitis is often nonspecific. However, a detailed neurologic examination may elicit focal deficits that not only support the diagnosis of encephalitis but also provide clues to the causative virus:

- **Myoclonus** – Myoclonus may be present in patients with subacute sclerosing panencephalitis following measles infection or less commonly with herpes simplex virus (HSV) type 1. Nonviral causes include prion disease and autoimmune encephalitis.
- **Movement disorders** – These may include hypo/dyskinesia or tremors and may be seen with Japanese encephalitis, West Nile virus, St. Louis encephalitis virus, and acute necrotizing encephalopathy due to influenza virus or other respiratory viruses.
- **Acute flaccid paralysis** – Acute flaccid paralysis, including a polio-like presentation, is suggestive of West Nile virus infection [38] or, in children, enteroviral infection (EV-D68 or EV-A71) [39].
- **Bulbar or ocular dysfunction** – These findings can be seen with human herpesvirus 6 (HHV-6) encephalitis as well as with listeria encephalitis and paraneoplastic encephalitides.
- **Psychosis or agitated delirium** – Psychiatric manifestations may suggest a diagnosis of rabies, HSV-1, or a noninfectious condition such as anti-NMDA receptor encephalitis or lupus cerebritis. Conversely, catatonia may be seen in a minority of rabies cases.

Extraneurologic findings that may support a particular pathogen include:

- **Parotitis** – Parotitis in an unvaccinated patient with mental status changes suggests the diagnosis of mumps encephalitis and can also be seen with lymphocytic choriomeningitis virus infection of the central nervous system (CNS). (See "Mumps".)

- **Rash** – Vesicular rash in a dermatomal pattern may suggest varicella-zoster virus (VZV) encephalitis. However, the absence of rash does not exclude VZV from consideration [40], and rash due to VZV may develop after the onset of neurologic symptoms. (See "Epidemiology, clinical manifestations, and diagnosis of herpes zoster".)

  A nonspecific morbilliform rash may be seen with measles or West Nile virus.

Imaging — In patients with encephalitis, imaging (eg, contrasted brain magnetic resonance imaging [MRI]) may demonstrate abnormal radiographic findings that support a diagnosis of encephalitis. These findings may have both diagnostic and prognostic significance and suggest specific etiologies. As examples:

- Temporal lobe involvement is strongly suggestive of HSV-1 encephalitis, although other herpes viruses (eg, HSV-2, VZV, HHV-6) can also produce this clinical picture [41,42]. (See "Herpes simplex virus type 1 encephalitis", section on 'Imaging studies'.)

- Involvement of the thalamus or basal ganglia may be observed in the setting of encephalitis due to West Nile virus, eastern equine encephalitis virus, or respiratory viral infections (eg, influenza) [43-46].

- Brainstem involvement can be seen with enteroviral encephalitis, particularly EV-A71. (See "Enterovirus and parechovirus infections: Clinical features, laboratory diagnosis, treatment, and prevention", section on 'Encephalitis'.)

- Cerebellar abnormalities may be found with Epstein-Barr virus (EBV) or VZV encephalitis.

- White matter changes consistent with demyelination may be seen with postinfectious encephalitides such as acute disseminated encephalomyelitis (ADEM) [8]. (See 'Viral versus postinfectious encephalitis' above.)

Electroencephalograph — Electroencephalography is often abnormal in acute encephalitis. Focality in the temporal lobe region is suggestive of HSV encephalitis, as noted above [47].

Cerebrospinal fluid findings — Examination of the CSF often indicates the presence of inflammation in the CNS, although a lack of inflammatory findings does not rule out encephalitis. In one large retrospective study, CSF white blood cell (WBC) count <5/microL was

observed in 19 percent of patients with encephalitis due to infection and 24 percent of patients with HSV-1 encephalitis [17,48].

The following findings are characteristic of viral CNS infections (table 3):

- Increased WBC count, but usually less than 250/microL. The differential shows a predominance of lymphocytes, although early infection may reveal a predominance of neutrophils. In the latter setting, a repeat CSF cell count eight hours later will generally show a shift from neutrophils to lymphocytes [49].
- Elevated protein concentration but usually less than 150 mg/dL.
- Usually normal glucose concentration (>50 percent of blood value), but moderately reduced values are occasionally seen with HSV, mumps, or some enteroviruses.
- Red cells are usually absent (in a nontraumatic tap); their presence in the appropriate clinical setting suggests HSV-1 infection or other necrotizing encephalitis [50].

CSF findings in viral encephalitis are generally quite different from those associated with bacterial meningitis (table 3). However, the spectrum of CSF findings in bacterial meningitis is wide, and exclusion of bacterial meningitis can be difficult. (See "Clinical features and diagnosis of acute bacterial meningitis in adults", section on 'Cerebrospinal fluid analysis' and 'Plus therapy for other pathogens' below.)

DIFFERENTIAL DIAGNOSIS

It is critical that the clinician considers a broad range of causes when evaluating patients with encephalitis. Only some of the causes are infectious, and not all infections are viral.

Several noninfectious etiologies can mimic central nervous system (CNS) infections. These include primary intracranial or metastatic tumors, adverse effects of medications, and autoimmune or paraneoplastic diseases, such as those associated with vasculitis or the anti-NMDA receptor (table 4). (See "Autoimmune (including paraneoplastic) encephalitis: Clinical features and diagnosis".)

Nonviral infectious etiologies to consider in a patient with suspected CNS infection include tickborne infections, such as Rocky Mountain spotted fever, listeriosis, neuroleptospirosis, syphilis, tuberculous meningitis, and fungal meningitis (eg, Coccidioides). Primary amoebic encephalitis due to *Naegleria fowleri* should be included in the differential diagnosis in patients with a history of freshwater exposure and rapidly progressive meningoencephalitis. (See

"Pathogenesis, clinical manifestations, and diagnosis of brain abscess" and "Central nervous system tuberculosis: An overview" and "Coccidioidal meningitis" and "Neurosyphilis".)

Knowledge of the patient's underlying immune status is also critical, since the differential diagnosis is even broader in immunocompromised hosts (eg, toxoplasmic encephalitis and cryptococcal meningitis). (See "Toxoplasmosis: Acute systemic disease" and "Epidemiology, clinical manifestations, and diagnosis of *Cryptococcus neoformans* meningoencephalitis in patients with HIV".)

DIAGNOSIS

Our approach — We suspect viral encephalitis in patients who meet clinical criteria for encephalitis as shown in the table (table 1). Given the large number of viruses that cause encephalitis, we recommend a two-tier approach. This includes universal testing for the most common and treatable pathogens, combined with additional testing in selected patients based on patient-specific characteristics.

Other nonviral infectious and autoimmune causes can produce a similar clinical picture of encephalitis (eg, syphilis, rickettsial infections, common bacterial causes of meningoencephalitis). We also perform diagnostic evaluation for nonviral infectious (eg, bacteria, fungi, mycobacteria) and noninfectious etiologies. (See 'Differential diagnosis' above.)

Initial testing

Core testing for all patients — For all adults with suspected viral encephalitis, we perform the following core tests:

- **Blood** – Core testing on blood includes human immunodeficiency virus (HIV) antibody test, treponemal antibodies (or rapid plasma reagin), and blood cultures. For all patients with symptom onset during late spring through late fall, we also test West Nile virus immunoglobulin (Ig)M.

- **Cerebrospinal fluid (CSF)** – Analysis of the CSF is required as an initial diagnostic step in patients with suspected viral encephalitis in the absence of contraindication (eg, increased intracranial pressure or coagulopathy). (See "Clinical features and diagnosis of acute bacterial meningitis in adults", section on 'Indications for CT scan before LP'.)

  CSF testing should include opening CSF pressure, cell count, glucose, protein, and bacterial culture. Routine viral testing of CSF should include polymerase chain reaction

(PCR) testing for herpes simplex virus (HSV)-1, HSV-2, varicella-zoster virus (VZV), and enteroviruses, either as single pathogen tests or through a multiplex panel. We also check CSF venereal disease research laboratory slide test (VDRL) and cryptococcal antigen. For all patients with symptom onset during late spring through late fall, we also test CSF West Nile virus IgM antibody.

- **Imaging and electroencephalogram** – For all patients with suspected viral encephalitis, we obtain a contrasted brain MRI. For patients with seizures or severely depressed mentation, we obtain an electroencephalogram to evaluate for nonconvulsive status epilepticus as a cause of altered mental status.

  The most important viral etiology to rule out in a patient with encephalitis is HSV-1 since this clinical entity is usually fatal if untreated, and delayed initiation antiviral of treatment is associated with poor neurologic outcomes. HSV PCR should be performed on CSF samples from all patients with encephalitis as atypical cases lacking focal temporal lobe involvement or pleocytosis may occur [17,48,51,52]. HSV PCR of CSF is both sensitive and specific, although may be negative early in the course of infection [53,54]. While awaiting confirmation, empiric therapy with intravenous acyclovir should be initiated. (See 'Empiric antimicrobial therapy' below.)

Given the ubiquity of West Nile virus, IgM antibody testing of blood and CSF is indicated in any adult presenting with encephalitis during a time of year when the mosquito vector might be present, typically late spring through late fall. (See "Clinical manifestations and diagnosis of West Nile virus infection", section on 'Diagnosis'.)

Enteroviruses are more commonly associated with viral meningitis but may cause encephalitis as well, particularly in patients with impaired B-cell immunity [55]. PCR testing on the CSF sample is the diagnostic test of choice. The pathogen can also be detected in stool and nasopharyngeal specimens; however, identification outside of the central nervous system (CNS) should be interpreted with caution as this may be an incidental finding. (See "Enterovirus and parechovirus infections: Clinical features, laboratory diagnosis, treatment, and prevention", section on 'Laboratory diagnosis' and "Molecular diagnosis of central nervous system infections", section on 'Other viruses'.)

We also test all adults with encephalitis for syphilis and *Cryptococcus*. Neurosyphilis is treatable, highly transmissible, and can mimic HSV with temporal lobe findings. *Cryptococcus* can occur in both immunocompromised and immunocompetent individuals and is fatal without treatment. (See "Syphilis: Screening and diagnostic testing", section on 'Diagnostic tests' and "Neurosyphilis", section on 'Diagnosis'.)

Additional testing for selected patients

- **Additional infectious pathogens** – We perform additional testing for other infectious pathogens (eg, arboviruses, rabies, Epstein-Barr virus [EBV]) in selected patients based on seasonality, geography, clinical features, and exposure history. (See 'Clues on physical examination' above and 'Epidemiologic clues' above.)

  As an example, respiratory symptoms suggest the possibility of respiratory viral or bacterial causes of encephalitis (eg, influenza, adenovirus, *Mycoplasma*). (See "Diagnosis, treatment, and prevention of adenovirus infection", section on 'Diagnostic approach' and "Seasonal influenza in adults: Clinical manifestations and diagnosis", section on 'Diagnosis of influenza' and "*Mycoplasma pneumoniae* infection in adults", section on 'Non-respiratory tract disease'.)

  Immunocompromised patients are at risk for other viral (eg, human herpesvirus 6 [HHV-6], cytomegalovirus, JC virus) and nonviral (eg, *Toxoplasma*) etiologies of encephalitis; these are discussed elsewhere. (See "Human herpesvirus 6 infection in hematopoietic cell transplant and CAR-T cell therapy recipients", section on 'Diagnosis' and "Progressive multifocal leukoencephalopathy (PML): Epidemiology, clinical manifestations, and diagnosis", section on 'Diagnosis' and "AIDS-related cytomegalovirus neurologic disease", section on 'Diagnosis'.)

  Lyme disease is an uncommon cause of encephalitis (as opposed to aseptic meningitis); thus, testing for Lyme is likely only indicated after negative initial workup in a patient who resides in a highly endemic area. For such patients, we check serum Lyme antibodies; if negative, we do not perform further CSF testing for Lyme disease. If serum Lyme antibody is positive, the approach to further testing is discussed elsewhere. (See "Diagnosis of Lyme disease", section on 'Need for additional testing'.)

- **Anti-NMDA receptor antibodies** – For patients who also present with new psychiatric or movement abnormalities, we include anti-NMDA receptor antibody testing (from both blood and CSF) with routine initial testing.

Testing for patients who do not improve — Further testing is required if the patient is not improving. In these cases, we repeat brain MRI and lumbar puncture to reassess for radiographic progression and worsening pleocytosis. Expanded testing for both infectious (eg, rabies) and noninfectious etiologies (eg, anti-NMDA receptor antibody tests from blood and CSF) is indicated. However, even with comprehensive testing, the etiology in many cases remains undefined [14,56]. (See 'Etiology often unidentified' above.)

Rabies should be considered in any patient with an undiagnosed progressive encephalitis, even in the absence of a compelling exposure history. Diagnosis of rabies requires testing of several specimens including saliva, nuchal skin biopsy, and CSF since the sensitivity of any single test is limited. Coordination with regional public health authorities is strongly recommended in these cases. (See "Clinical manifestations and diagnosis of rabies", section on 'Diagnosis'.)

Test methods

Molecular testing

- **PCR** – The sensitivity of PCR testing varies by pathogen. As examples, for HSV and enterovirus infections of the CNS, CSF PCR is the most sensitive test. For West Nile virus, PCR sensitivity varies by immune status; in immunocompetent patients, West Nile virus CSF PCR testing is insensitive, and CSF IgM antibody is the preferred diagnostic modality [57]. By contrast, for immunocompromised patients (particularly those treated with B-cell depleting agents [eg, rituximab]), viral persistence in the CSF renders PCR more sensitive than serology [58]. The role of PCR testing by suspected pathogen is outlined further in the table, although not all tests in this table are recommended for the workup of encephalitis (table 5).

  Caution should be exercised in interpreting a positive PCR result for viruses that may reactivate in the setting of other infections, such as EBV, or may be chromosomally integrated, such as HHV-6 [7]. These issues and other caveats to testing and interpretation of CSF molecular testing are discussed further elsewhere. (See "Molecular diagnosis of central nervous system infections", section on 'Caveats to testing and interpretation'.)

- **Sequencing-based tests** – Meta-genomic next generation sequencing (NGS) of CSF allows unbiased amplification of deoxyribonucleic acid (DNA) and ribonucleic acid (RNA), allowing identification of pathogens that may not be clinically suspected [59-61]. While NGS does not replace traditional diagnostic testing, this modality should be considered in hospitalized patients who fail to improve and in whom initial tests are unrevealing [7].

The role of molecular diagnostics for viral and other causes of encephalitis are discussed further elsewhere. (See "Molecular diagnosis of central nervous system infections".)

Serology — Serologic testing remains the preferred test for arboviral encephalitis. Definitive diagnosis of acute infection requires demonstration of a fourfold rise in titer or seroconversion. Convalescent serology should be obtained no sooner than two weeks after

the onset of the clinical illness. As an example, the presence of IgM antibodies in a single serum provides presumptive evidence of St. Louis encephalitis; however, a fourfold change between appropriately timed acute and convalescent sera is considered diagnostic.

For West Nile virus, finding IgM antibodies in the serum or CSF sample is suggestive but not diagnostic of West Nile virus infection as IgM antibody may be detectable for >5 years after infection [62]. Additionally, there may be cross-reactivity with West Nile virus and other flaviviruses, such as St. Louis encephalitis virus or Japanese encephalitis virus [63]. (See "Clinical manifestations and diagnosis of West Nile virus infection", section on 'Diagnosis' and "Arthropod-borne encephalitides", section on 'Specific viruses'.)

Serology may also be helpful in obtaining evidence for primary EBV infection, a rare cause of meningoencephalitis, particularly as amplification of EBV in CSF may simply reflect reactivation of latent virus.

Other tests with limited role

- **Culture** – CSF viral culture is insensitive and has a limited role in the evaluation of encephalitis. Of 22,394 CSF samples submitted for viral culture, a pathogen was grown in <6 percent, with enteroviruses accounting for >98 percent of isolates [64].

  Given the poor sensitivity, in almost all cases, CSF PCR testing has replaced viral culture in the evaluation of encephalitis.

- **Brain biopsy** – Brain biopsy can be considered when no diagnosis is identified by conventional testing. This procedure may identify a viral or nonviral (potentially treatable) etiology. In a contemporary study, brain biopsy identified a pathogen in 31 percent of patients with undiagnosed encephalitis and was particularly high yield in immunocompromised patients [65], Notably, metagenomic NGS performed on brain tissue may have a higher yield than on CSF [66,67]. Multidisciplinary preoperative discussion regarding anatomic sites of sampling, optimal specimen collection (both fixed and fresh-frozen tissue), and testing prioritization may optimize the diagnostic yield of this invasive procedure.

MANAGEMENT

Empiric antimicrobial therapy

Antiviral therapy — For all patients with suspected viral encephalitis, we initiate empiric treatment for herpes simplex virus (HSV)-1 infection with acyclovir (10 mg/kg intravenously

every eight hours assuming normal kidney function) as soon as possible [68]. Empiric acyclovir treatment is warranted in all patients with encephalitis, given the frequency of HSV-1 as a cause of encephalitis and the relationship between early treatment and favorable outcome [69] (see "Herpes simplex virus type 1 encephalitis"). Acyclovir is also active against varicella-zoster virus (VZV).

The approach to continuing acyclovir therapy depends on the results of cerebrospinal fluid (CSF) polymerase chain reaction (PCR) testing and the clinical suspicion for HSV encephalitis:

- For patients with a positive CSF PCR confirming HSV, treatment is discussed elsewhere (see "Herpes simplex virus type 1 encephalitis", section on 'Treatment'). For patients with a positive PCR for VZV, we administer 10 to 15 mg/kg IV acyclovir every eight hours (with adjustments for impaired kidney function) for 14 days.

- For patients with a negative CSF PCR for HSV and VZV, we discontinue acyclovir if an alternative diagnosis is made (eg, West Nile virus). For patients without a confirmed alternative diagnosis, the approach to discontinuation of empiric acyclovir depends on clinical suspicion and is discussed elsewhere. (See "Herpes simplex virus type 1 encephalitis", section on 'Discontinuation of therapy based on PCR results'.)

Plus therapy for other pathogens — Since a number of other treatable illnesses may present in a similar manner to viral central nervous system (CNS) infections, initial empiric therapy for alternative diagnoses (particularly bacterial and/or tickborne meningoencephalitis, which are both common and treatable) is essential (table 4) [6,7,70,71].

- **Common causes of bacterial meningitis** – Given the overlap between meningitis and encephalitis and the desire to avoid delayed treatment, we initially administer empiric therapy for bacterial meningitis in all patients who present with encephalitis. (See "Initial therapy and prognosis of community-acquired bacterial meningitis in adults", section on 'Choice of regimen'.)

  In general, we discontinue antibiotic therapy in patients with negative CSF bacterial cultures at 48 to 72 hours or a negative CSF multiplex molecular panel test for common causes of bacterial meningitis, unless other features suggest bacterial meningitis (eg, neutrophilic pleocytosis, antibiotic pretreatment).

  For patients with encephalitis, it is initially difficult to exclude bacterial meningitis based only on CSF parameters. Although CSF findings in viral encephalitis are generally quite different from those associated with bacterial meningitis (table 3), the spectrum of CSF

values in bacterial meningitis is so wide that the absence of one or more of these findings is of little value. This was illustrated in a review of 296 episodes of community-acquired bacterial meningitis; 50 percent had a CSF glucose above 40 mg/dL (2.2 mmol/L), 44 percent had a CSF protein below 200 mg/dL, and 13 percent had a CSF white blood cell (WBC) count below 100/microL [72]. (See "Clinical features and diagnosis of acute bacterial meningitis in adults", section on 'Cerebrospinal fluid analysis'.)

- **Tick-borne infections** – For patients at risk for treatable tick-borne infections (eg, rickettsial infections [*Ehrlichia*, Rocky Mountain spotted fever], Lyme), we include empiric doxycycline in the initial antimicrobial regimen [7]. Such patients include those with tick exposure during spring through fall in endemic regions. If an alternate diagnosis is established or the patient has no improvement after three days of therapy, we stop doxycycline.

Increased intracranial pressure — Symptoms and signs of increased intracranial pressure (ICP) include headache, vomiting, and a decreased level of consciousness. Serial ICP monitoring should be continued in a patient with documented elevated ICP since this parameter has been associated with a negative prognosis in encephalitis [73]. Relieving elevated ICP may decrease secondary brain injury while the patient is responding to anti-infective therapy [74]. Whether any of these interventions have a positive impact on outcome has not been determined in prospective clinical trials. Various strategies to reduce ICP are discussed elsewhere. (See "Evaluation and management of elevated intracranial pressure in adults", section on 'General management'.)

"Standard" therapeutic interventions for lowering CSF pressure (eg, glucocorticoids, mannitol) have been used in this setting; use of glucocorticoids in patients with encephalitis is discussed further elsewhere. (See 'Role of glucocorticoids' below.)

Role of glucocorticoids — For patients with encephalitis, we avoid empiric glucocorticoids in the absence of other indications (eg, increased ICP); data do not support a benefit, and there are concerns regarding potential harm.

For patients suspected or confirmed autoimmune encephalitis, glucocorticoids are often used. (See "Autoimmune (including paraneoplastic) encephalitis: Management", section on 'Glucocorticoids'.)

For patients with viral encephalitis, studies do not show improved outcomes with use of glucocorticoids. A meta-analysis of 50 studies did not identify a beneficial treatment effect with adjuvant corticosteroids among patients with viral encephalitis [75] (see "Initial therapy and prognosis of community-acquired bacterial meningitis in adults", section on 'Adjunctive

dexamethasone'). A prospective study of 94 adults with HSV encephalitis demonstrated similar cognitive outcomes at six months and adverse events in patients treated with or without dexamethasone [76]. However, use of glucocorticoids in patients with West Nile virus neuroinvasive disease has been associated with increased mortality [77].

In patients with encephalitis and negative testing for infectious causes, if there is high clinical suspicion for an autoimmune etiology, empiric glucocorticoids may be used on a case-by-case basis. We manage these cases with multidisciplinary input from infectious diseases and neurology specialists.

PROGNOSIS

The prognosis of viral encephalitis varies based on the pathogen and the host. For instance, rabies is almost uniformly fatal, whereas enteroviral encephalitis is typically associated with full recovery.

There are limited data on long-term outcomes with infectious encephalitis. A retrospective analysis of 41 studies involving more than 3000 adults with viral encephalitis reported that the majority of patients had residual neurologic or cognitive impairment at the time of hospital discharge [78]. Studies that included long-term follow-up found clinical improvement can occur up to several years after infection, suggesting that aggressive rehabilitation efforts may be advantageous [79].

SOCIETY GUIDELINE LINKS

Links to society and government-sponsored guidelines from selected countries and regions around the world are provided separately. (See "Society guideline links: Infectious encephalitis".)

INFORMATION FOR PATIENTS

UpToDate offers two types of patient education materials, "The Basics" and "Beyond the Basics." The Basics patient education pieces are written in plain language, at the 5^{th} to 6^{th} grade reading level, and they answer the four or five key questions a patient might have about a given condition. These articles are best for patients who want a general overview and who prefer short, easy-to-read materials. Beyond the Basics patient education pieces are longer, more sophisticated, and more detailed. These articles are written at the 10^{th} to 12^{th} grade

reading level and are best for patients who want in-depth information and are comfortable with some medical jargon.

Here are the patient education articles that are relevant to this topic. We encourage you to print or e-mail these topics to your patients. (You can also locate patient education articles on a variety of subjects by searching on "patient info" and the keyword(s) of interest.)

- Basics topics (see "Patient education: Encephalitis (The Basics)")

SUMMARY AND RECOMMENDATIONS

- **Epidemiology** – The most common viral causes of encephalitis in the United States include herpes simplex virus (HSV) type 1, varicella-zoster virus (VZV), enteroviruses, and West Nile virus. Other viral causes of encephalitis are associated with specific geographic, seasonal, or animal exposures. (See 'Epidemiology' above.)

- **Clinical presentation** – Clinical criteria for encephalitis are shown in the table (table 1). Altered mental status is a requisite finding, while other signs or symptoms (fever, seizures, and meningeal signs) are variably present. The term meningoencephalitis is used to recognize that meningitis and encephalitis frequently overlap. (See 'Meningitis versus encephalitis' above and 'Signs and symptoms' above.)

- **Diagnosis** – Given the large number of viral, other infectious, and noninfectious etiologies of encephalitis, we recommend a two-tier approach to initial testing. (See 'Diagnosis' above.)

  - **Core testing for all patients** – For all patients, we obtain a contrasted brain MRI to assess for findings that support a diagnosis and/or suggest specific etiologies (eg, temporal lobe involvement with HSV-1). (See 'Imaging' above.)

    In adults, we also test for the most common and treatable causes of encephalitis:

    - Blood tests include HIV antibody, syphilis testing, and blood cultures.

    - Cerebrospinal fluid (CSF) tests include opening pressure, cell count, glucose, protein, bacterial culture, viral polymerase chain reaction tests (PCRs) for HSV-1, HSV-2, VZV, and enteroviruses, venereal disease research laboratory slide test (VDRL), and cryptococcal antigen.

    - For all patients with symptom onset during spring through fall, we also test West Nile virus IgM from blood and CSF.

For patients with seizures or severely depressed mentation, we obtain an electroencephalogram to evaluate for nonconvulsive status epilepticus.

- **Additional risk-based testing for selected patients** – We perform additional testing for other infectious etiologies (eg, arboviruses, rabies, Epstein-Barr virus [EBV]) and noninfectious (eg, autoimmune) etiologies in selected patients based on seasonality, geography, clinical features, and exposure history. (See 'Additional testing for selected patients' above and 'Epidemiologic clues' above and 'Clues on physical examination' above.)

- **Empiric therapy** – For all patients with encephalitis, empiric therapy consists of intravenous acyclovir (for activity against HSV and VZV) and antibiotics with activity against common causes of bacterial meningitis. For those with tick exposure during spring through fall in endemic regions, we also include empiric doxycycline for activity against tick-borne infections (eg, rickettsial infections). (See 'Empiric antimicrobial therapy' above and "Herpes simplex virus type 1 encephalitis", section on 'Treatment'.)

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